CLAIMS

1. Use of peptide antagonists at glutamate receptors for the manufacture of a medicament to influence the glutamate-receptor-controlled cells.

2. Use of peptide antagonists at NMDA receptors for the manufacture of a medicament to influence the NMDA-receptor-controlled cells.

3. Use according to claim 2 in which the medicament prevents NMDA-receptor-mediated excitatory effects such as release of neurotransmitter or peptide as well as toxic effects resulting in cell injury or death.

4. Use according to any of claims 1 to 3 in which the cells are neurons or glial cells in the central nervous system.

5. Use according to any of claims 1 to 4 in which the medicament comprises glutamic acid-terminating peptides.

6. Use according to any of claims 1 to 5 in which the antagonist is chosen among (1-5)GnRH, (1-3)IGF-I, (1-37)GRF and C-peptide of insulin.

7. Use according to any of claims 1 to 6 in which the medicament influence GnRH secretion.

8. Use according to any of claims 1 to 7 for the treatment of acute or chronic disorders of the central nervous system.

9. Use according to any of claims 1 to 7 for the treatment of hypoxic, ischemic and metabolic brain disorders such as stroke and hypoglycaemia, traumatic, radiation-induced or inflammatory injuries to the brain and chronic degenerative states.

10. Use according to any of claims 1 to 9 for the treatment of children during the perinatal period and infancy.

11. Use according to any of claims 1 to 10 in which the medicament comprises (1-3) IGF-I.

12. Use according to any of claims 1 to 11 in which the medicament is administered systemically.

13. Use according to any of claims 1 to 11 in which the medicament is administered locally.

14. Method for influence on glutamate-receptor-controlled cells by administration of peptide antagonists at glutamate receptors.

15. Method for influence on NMDA-receptor-controlled cells by administration of peptide antagonists at NMDA receptors.

16. Method according to claim 15 for preventing NMDA-receptor mediated excitatory effects such as release of neurotransmitter or peptide as well as toxic effects resulting in cell injury or death.

17. Method according to any of claims 14 to 16 for influence on the function of neurons or glial cells in the central nervous system.

18. Method according to any of claims 14 to 17 in which the antagonists at NMDA receptors comprises glutamic acid-terminating peptides.

19. Method according to any of claim 14 to 18 in which the antagonist is chosen among (1-5)GnRH, (1-3)IGF-I, (1-37)GRF and C-peptide of insulin.

20. Method according to any of claim 14 to 19 for influencing the GnRH secretion.

21. Method according to any of claims 14 to 120 for the treatment of acute or chronic disorders of the central nervous system.

22. Method according to any of claims 14 to 20 for the treatment of hypoxic, ischemic and metabolic brain disorders such as stroke and hypoglycaemia, traumatic, radiation-induced or inflammatory injuries to the brain and chronic degenerative states.

23. Method according to any of claims 14 to 21 for the treatment of children during the perinatal period and infancy.

24. Method according to any of claims 14 to 22 in which a medicament is administered which comprises (1-3) IGF-I.

25. Method according to any of claims 14 to 23 in which a medicament is administered systemically.

26. Method according to any of claims 14 to 23 in which a medicament is administered locally.